In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 18-1012V

(to be published)

ANA SANCHEZ,	*	
	*	Chief Special Master Corcoran
Petitioner,	*	Filed: March 11, 2022
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Lawrence R. Cohen, Saltz Mongeluzzi & Bendesky, Philadelphia, PA, for Petitioner.

Tyler King, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On July 13, 2018, Ana Sanchez filed this action seeking compensation under the National Vaccine Injury Compensation Program (the "Program").[2] ECF No. 1. Petitioner alleges that a tetanus, diphtheria, and acellular pertussis ("Tdap") vaccine she received on March 17, 2016, caused her to incur chronic inflammatory demyelinating polyneuropathy ("CIDP"). An entitlement hearing in the matter was held in Washington, D.C., on September 15, 2021.

Having reviewed the record, all expert reports and associated literature, and listened to those witnesses and experts who testified at the hearing, I hereby deny an entitlement award. As

[1] This Decision will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). **This means the Decision will be available to anyone with access to the internet**. As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the published Ruling's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the entire Decision will be available to the public in its current form. *Id.*

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

discussed in greater detail below, Petitioner has not preponderantly established that the Tdap vaccine can cause CIDP, or that it did so to her in the relevant timeframe.

I. Fact History

Circumstances of Vaccination

Ms. Sanchez was born on August 22, 1984. Ex. 1 at 3. She is the mother of three children. *Id*. Prior to the vaccination at issue, her medical history was significant for hypertension, chronic rhinitis, fibromyalgia, and fatigue.[3] *Id.* at 27. On March 17, 2016, she received the Tdap vaccine from San Antonio Family Physicians in San Antonio, Texas when she was 31 years old (and at that time pregnant). *Id.* at 3.

Petitioner gave birth on April 28, 2016, and subsequently visited her gynecologist, Michelle Harden, M.D., on May 3, 2016, reportedly for swelling, numbness in her feet and legs, and elevated blood pressure. Ex. 12 at 13; Tr. at 19. It was now seven weeks from vaccination. Dr. Harden referred Petitioner to the Westover Hills emergency room ("ER"), where she complained of hypertension, headaches, neck pain, blurred vision, and edema to lower extremities, but no fever or dysuria. Ex. 8 at 4, 9. Petitioner, however, has testified that her main complaint at this time was numbness—despite the fact that the medical record focuses on hypertension and high blood pressure. Tr. at 13-14. Petitioner specifically noted at this time (as reflected in the medical record) that "she began to feel 'weird' three days ago [on May 1, 2016]." Ex. 8 at 9. The treating physician observed that Petitioner had hypertension but did not believe her symptoms reflected preeclampsia.[4] *Id.* at 10. Petitioner was given medication to reduce her blood pressure. *Id.*

Notably, the medical records in this case do not record complaints of neurologic symptoms as of this timeframe. Petitioner, however, has alleged that the symptoms that later were diagnosed as CIDP had already begun—as early as the middle of April. Those allegations are discussed in more detail below.

Initial Neurologic Symptoms

About one week later, on May 6, 2016, Petitioner visited her primary care physician, Eric Bernstein, M.D., for an ultrasound. Ex. 11 at 7. The radiology report record contains Dr.

[3] In addition, in 2015 Petitioner was involved in a car accident (although the exact date is not recorded) and visited a chiropractor on several occasions for treatment. On her first such visit (April 23, 2015), Petitioner complained of pain in her neck, right trap shoulder, right wrist, back, left buttock and left thigh and rated her pain scale as a 10 out of 10. Ex. 5 at 11. Other follow-up visits, involving conservative chiropractic treatment, occurred in 2015 on April 27; May 1, 4, and 15; June 2, 4, 12, 17, and 29; July 6 and 14. *Id.* at 13, 15, 17, 19, 21, 24–25, 27, 29, 31. Petitioner was cross-examined at trial about the accident, but denied her post-vaccination symptoms were associated with it. Tr. at 28, 30–31. I do not conclude herein that it has been preponderantly demonstrated that this accident was in any way related to Petitioner's subsequent development of CIDP.

[4] Preeclampsia is defined as "a complication of pregnancy characterized by hypertension, edema, and/or proteinuria; when convulsions and coma are associated." *See Dorland's Illustrated Medical Dictionary* (33d ed. 2020) at 1486.

Bernstein's speculation that her symptoms could be caused by her thyroid. *Id.* There is a subsequent treatment gap in the record, with another month passing until Petitioner saw Dr. Bernstein again (on June 7, 2016), at which time she complained of pain and weakness in her hands and legs, which she reported had begun about three weeks prior (or in the middle of May). Ex. 1 at 24. A neurological exam now revealed decreased sensation bilaterally over her hands and feet but normal reflexes and gait. *Id.* at 25–26. These symptoms, assuming they started in mid-May, would have presented six to seven weeks after the vaccination in question.

Dr. Bernstein referred Ms. Sanchez to neurologist Jennifer Sharron, M.D. Ex. 1 at 26. Petitioner saw Dr. Sharron on June 21, 2016, at Alamo Neurology Consultants. Ex. 7 at 89.[5] She now reported to Dr. Sharron that a few days after giving birth, she had high blood pressure, palpitations, and felt like something popped in her neck. *Id.* The medical records from this visit also set forth that Petitioner complained of numbness and weakness in her hands that started in mid-May, which had progressed to her toes and feet and into her arms and legs. *Id.* This caused difficulty walking and carrying her young children. *Id.* She also reported that water caused the numbness and weakness to worsen. *Id.* A neurologic exam performed at this time was "notable for symmetric weakness more distal and proximal. She ha[d] impaired temperature sensation: intact light touch, proprioception, and vibration. She ha[d] 1 + DTRs in BUE, but absent in patellar and Achilles." *Id.* at 90. Dr. Sharron diagnosed Petitioner with Guillain-Barré syndrome ("GBS") and ordered a complete diagnostic workup, including labs, MRIs, lumbar puncture, an electromyogram ("EMG") and nerve conduction study ("NCS"), and IVIG for treatment. *Id*.

Evolution of Treatment

On July 19, 2016, Petitioner had a follow-up appointment with Dr. Sharron for further treatment of paresthesias. Ex. 7 at 87.[6] Petitioner had completed a five-day course of IVIG two weeks prior (beginning on June 28, 2016) with "significant improvement in her weakness," although she felt like she was still struggling with her strength. *Id.* at 87–88. A neurologic exam showed normal strength, normal sensation, and trace reflexes. *Id.* Dr. Sharron again diagnosed Petitioner with GBS, noting a lumbar puncture that revealed elevated CSF protein with normal cell count. *Id.* at 88. Dr. Sharron then referred the Petitioner to physical and occupational therapy and prescribed Gabapentin, a drug used to treat neuropathic conditions. *Id.*

On July 29, 2016, Petitioner returned to Dr. Sharron for a follow-up appointment, reporting increased weakness and numbness in her face. Ex. 7 at 85. Dr. Sharron felt "giveway weakness throughout" Petitioner's strength test (meaning weakness without an identifiable neurologic explanation) but decided to go ahead with "a maintenance dose of IVIG in case this might be a CIDP picture." *Id.* At this time, Petitioner was awaiting an EMG/NCS with a neuromuscular specialist, and Dr. Sharron referred Petitioner for home physical therapy. *Id.* The EMG and NCS

[5] Also documented in Ex. 2 at 18 and Ex. 11 at 10.

[6] Also documented in Ex. 2 at 16.

were performed the following month (on August 18, 2016) and revealed evidence of an "acquired sensorimotor demyelinating polyneuropathy of moderate severity." *Id.* at 42–44.

On August 25, 2016, Petitioner returned to Dr. Sharron for a follow-up appointment for her paresthesias. Ex. 7 at 83.[7] Dr. Sharron noted that Petitioner had received a second round of IVIG on August 3, 2016, and "felt better for a week, but then felt worsening weakness." *Id.* After a review of Petitioner's medical history and EMG/NCS results, Dr. Sharron's new working diagnosis was CIDP. *Id.* Upon examination, Petitioner showed normal sensation to light touch and normal reflexes. *Id.* Dr. Sharron prescribed Petitioner with IVIG treatments every two weeks and a continuation of Gabapentin. *Id.* at 83–84.

Several months later, on November 28, 2016, Petitioner returned to Dr. Sharron, complaining of dizziness, balance problems, and chest pains. Ex. 7 at 81.[8] Dr. Sharron noted Petitioner's plateau with IVIG and subsequent rashes (reflecting a side effect of IVIG). *Id.* An exam showed distal upper and lower extremity weakness, with strength at 3+/5. *Id.* Dr. Sharron suggested starting steroids, but Petitioner declined because she was breastfeeding. *Id.* at 81–82. Dr. Sharron increased Petitioner's Gabapentin and prescribed a continuation of physical and occupational therapy and IVIG. *Id.*

Embrace of CIDP as Proper Diagnosis

By 2017, additional evidence of Petitioner's neurologic injury was accumulating, although treaters did not always concur as to the proper diagnosis. On January 20, 2017, Petitioner saw Dr. Sharron to discuss disability forms. Ex. 7 at 79. At this time, she reported that she still felt "weak, unbalanced," and that her symptoms limited her in daily activities. *Id.* Upon further examination, Petitioner showed normal bilateral upper and lower extremity to light touch. *Id.* Dr. Sharron diagnosed Petitioner with CIDP and depression. *Id.* at 79–80. Dr. Sharron recommended a treatment plan of oral steroids and antidepressant medication. *Id.* She was to follow up in six weeks. *Id.* at 80. Petitioner later reported that after about five days on Prednisone, she stopped taking it due to dizziness and other side effects. *Id.* at 6.

Two months later, on March 30, 2017, Petitioner had a routine exam with Dr. Bernstein, where she made no mention of CIDP. Ex. 1 at 18–23.[9] An examination showed normal strength, sensation, and DTRs. *Id.* at 23. On July 19, 2017, Petitioner returned to Dr. Sharron for a follow-up appointment, where Dr. Sharron referred Petitioner to a neuromuscular specialist for a second

[7] Also documented in Ex. 2 at 12.

[8] Also documented in Ex. 2 at 10.

[9] Also documented in Ex. 2 at 6.

opinion. Ex. 7 at 78. Dr. Sharron also noted Petitioner's strength exam again showed giveway weakness. *Id.*

On September 6, 2017, Petitioner saw neuromuscular specialist Patrick Grogan, M.D., for a second opinion. Ex. 7 at 6–8, 55–57. After reviewing her medical history, Dr. Grogan assessed Petitioner as having peripheral demyelinating neuropathy, possibly GBS, with onset around May 2016 but which had subsequently resolved, leaving her now dealing with a separate chronic pain disorder like fibromyalgia or chronic fatigue. *Id.* at 7. Dr. Grogan rejected CIDP as the proper diagnosis because he would have "expected deterioration and objective examination abnormalities without any treatment for many months," but did not see it in Petitioner, adding that the abnormal EMG and NCS from August 2016 could reflect "residual [electrodiagnostic] abnormalities from the initial GBS," or a "hereditary neuropathy." *Id.* Dr. Grogan noted that Petitioner's examination was (as before) "severely limited by giveway weakness/poor effort." *Id.*

On October 12, 2017, Petitioner returned to Dr. Sharron for a follow-up appointment regarding Petitioner's ongoing paresthesias. Ex. 1 at 54.[10] An examination revealed normal sensation to light touch, and reduced strength in the wrists, hands, and legs. *Id.* Dr. Sharron's assessments were CIDP, neuropathic pain, and chronic fatigue. *Id.* Dr. Sharron ordered another EMG/NCS test and a gave Petitioner a referral to a pain specialist. *Id.*

Going forward, Ms. Sanchez has continued to seek treatment for neurologic symptoms, but has not consistently displayed strength or reflex issues. *See, e.g.,* Ex. 9 at 16, 19-20 (January 2018 visit to new neurologist). Exams from neurologist Ratna Bhavaraju-Sanka, M.D., have also revealed more instances of giveway weakness and inconsistent sensory problems, leading treaters to propose simply that Petitioner suffers from an unspecified form of neuropathy. Ex. 9 at 29, 31–32 (April 2018 neurologist visit). Petitioner asserts, however, that she continues to suffer from weakness in her extremities, balance issues, fatigue, leg cramping, difficulty sleeping, burning sensations in her hands, trouble gripping objects, difficulty swallowing, and hand tremors. Ex. 3 at 1–2.

II. Witness Testimony

A. *Ana Sanchez*

Petitioner was the only fact witness to testify. *See generally* Tr. at 5-32. She stated that she had no medical conditions of significance prior to giving birth to her third baby on April 28, 2016.[11] *Id.* at 6. However, towards the end of her pregnancy (about two to one and a half weeks before giving birth), she started to experience numbness in her feet. *Id.* at 6–7, 25. She informed

[10] Also documented in Ex. 2 at 4.

[11] Ms. Sanchez was discharged the next day on April 29, 2016. *Id.* at 7.

her doctor, who attributed it to the baby. *Id.* at 7. It was thus in mid-April, Petitioner contends, that her neurologic symptoms first manifested.

About two to three days after delivery, Ms. Sanchez was experiencing more numbness in her feet and swelling in her legs, so she visited the doctor. Tr. at 7–8. Specifically, on May 3, 2016, she saw, Dr. Harden, who had helped deliver her baby a few days prior. *Id.* at 8–9. Although the note from that visit indicates that Petitioner was complaining only of swelling in her lower extremities, Petitioner consistently during her testimony disputed the record's accuracy. *Id.* at 10, 29–30, 31. She instead maintained that the reason she had called the doctor was because of numbness in her feet and legs. *Id.* at 10, 24. Subsequently, Petitioner was referred to the ER for what she believed was treatment for hypertension. *Id.* at 10–11. The record of the neurology referral from Dr. Harden also fails to address or mention numbing or tingling, but again Petitioner denied the record's accuracy. *Id.* at 25–26; Ex. 12 at 13.

That same day, Petitioner visited the Westover Hills ER, where she was evaluated for hypertension. Tr. at 13. The ER record noted that "[patient] states she began to feel 'weird' three days ago," and Petitioner recalled in her testimony that the weird feeling was due to the numbness. *Id.*; Ex. 8 at 9. Other notes from this visit, however, also mentioned Petitioner's chest pain, headache, blurry vision, edema to lower extremities, but she emphasized telling the doctors about her numbness. Tr. at 14. She recalled getting medication for her headache and high blood pressure. *Id.* at 14–15. Following this ER visit, Petitioner testified, the numbness began spreading in her feet, legs, and hands, and she was experiencing an overall weakness in her legs that made it difficult to walk or hold her baby. *Id.* at 15–16.

Petitioner then discussed a few different doctor visits from various points in her subsequent treatment, with the aim of bulwarking her contention that she had experienced an April onset of neurologic symptoms. For example, she testified about a neuromuscular consult on January 24, 2018, where a note from this visit discussed preeclampsia, high blood pressure, and numbness in her legs as of April 2016. *Id.* at 16–18. She had also mentioned onset beginning around the time of her giving birth at a neurology consult on September 6, 2017. *Id.* at 18–19; Ex. 7 at 6–8, 55–57. And she noted in the record a visit to UT Health San Antonio on August 18, 2016, which documented that she was "presenting with distal/proximal weakness and distal numbness affecting 4 limbs, started within a week of delivery in April 2016." *Id.* at 19; Ex. 9 at 71. She viewed this as consistent with her recollection that her symptoms began mid-April. Tr. at 19–20. In contrast, the record from a June 21, 2016 visit to Dr. Sharron reports her neurologic symptoms only began in mid-May, but Petitioner disputed the record's accuracy. *Id.* 20; Ex. 9 at 89.

Petitioner otherwise provided an overview summary of her symptoms progression leading to the CIDP diagnosis. Thus, on May 6, 2016, Petitioner noted that she reached out to her primary care doctor who originally thought the symptoms were due to her thyroid, but eventually referred her to Dr. Sharron. Tr. at 21; Ex. 11 at 7. By June, when the visit with Dr. Sharron occurred, it was now proposed that Petitioner had GBS and needed IVIG treatment for it. Tr. at 21–22; Ex. 7 at 89–

90. Petitioner did not, however, find this immune-modulating treatment effective, however, noting that while it had some initial positive impact, the relief it provided diminished over time. Tr. at 22. After Petitioner had EMG and NCS testing completed, Dr. Sharron changed Petitioner's diagnosis from GBS to CIDP. *Id.* Although Petitioner has not had a differing diagnosis since, she indicated that the medication for CIDP had not helped, and that her symptoms of numbness, weakness, and pain continue to this day. *Id.* at 22–23.

B. *Petitioner's Expert - Frederick Nahm, M.D., Ph.D.*

Dr. Nahm, a clinical neurologist with 17 years of experience in acute neurological emergencies and outpatient and rehabilitation settings, prepared three written reports and testified for Petitioner in support of the contention that the Tdap vaccine can cause CIDP, and that it did so in this case. *See generally* Tr. at 33–96, 129–138. Report, dated September 8, 2020, filed as Ex. P13 (ECF No. 31-1) ("Nahm First Rep."); Report, dated June 6, 2021, filed as Ex. P18 (ECF No. 42-1) ("Nahm Second Rep."); Report, dated August 13, 2021, filed as Ex. P16 (ECF No. 50-1) ("Nahm Third Rep.").

Dr. Nahm received his Bachelor of Science from the University of Michigan, his Master of Science and Ph.D. from the University of California, San Diego, and his medical degree from the University of Michigan Medical School. CV, filed as Exhibit P14 on September 30, 2020 (ECF No. 32-1) ("Nahm CV") at 1; Tr. at 33–35. He also completed his neurology residency at Harvard Medical School. Tr. at 34. Dr. Nahm currently serves as the founder of the private neurology practice NeuroCare Health, P.C. and is the Intraoperative NeuroMonitoring Physician at NuVasive Clinical Services. Nahm CV at 1.

Over the course of his career, Dr. Nahm has treated, evaluated, and diagnosed over 100 patients with immune-mediated neuropathies such as GBS and CIDP. Tr. at 36. He is licensed to practice medicine in over 23 states and is board certified by the American Board of Psychiatry and Neurology and the American Board of Electrodiagnostic Medicine. Nahm CV at 1; Tr. at 37. Dr. Nahm has averred that he has treated and diagnosed people with CIDP in particular, and has first-hand knowledge of both the clinical features and the clinical course related to this condition. Nahm First Rep. at 1. He also treats about five to seven GBS patients a year. Tr. at 81–82.

One aspect of Dr. Nahm's opinion was devoted to addressing the veracity of Ms. Sanchez's CIDP diagnosis, which he favored over GBS. Tr. 46–47, 130; Nahm First Rep. at 16. Dr. Nahm defined GBS as a prototypical immune-mediated polyneuropathy, also referred to as acute inflammatory demyelinating polyneuropathy ("AIDP"). Nahm First Rep. at 16. CIDP, also an immune-mediated demyelinating neuropathy, affects both large and small fiber peripheral nerves, resulting in symptoms of numbness, tingling, weakness, imbalance, loss of coordination and pain. *Id.* at 17. It can, he added, be thought of as a chronic form of AIDP, with a course often exceeding two months. *Id.*; Tr. at 51-52; M. Fadia et al., *Immune-Mediated Neuropathies, Current Treatment Options Neurology* 1, 1 (2019), filed as Ex. P15c on Sept. 30, 2020 (ECF No. 32-2); R.

Hanewinckel et al., *Chapter 15 Peripheral Neuropathies*, Handbook Clinical Neurology 263, 265 (2016), filed as Ex. P22 on June 11, 2021 (ECF No. 42-7) ("Hanewinckel"). Hanewinckel specifically points to a clinical distinction in terms of temporal progression, noting that CIDP involves a steady, chronic progression over many weeks, whereas GBS patients experience a precipitous/acute kind of weakness within one to two weeks after onset that usually reaches nadir then gradually shows improvement over time. Tr. at 52, 82–83; Hanewinckel at 365.

Although CIDP's causes are largely not understood, a prevailing explanatory theory is that it develops in response to some environmental antigen that then triggers an immune-oriented inflammatory response, leading to nerve and myelin injury. Nahm First Rep. at 17, 19; S. Kuwabara, (2018). *Chronic Inflammatory Demyelinating Polyneuropathy: The Spectrum and Immunopathogenesis Deciphered by Electrophysiology and Neuroimaging*, Clinical & Experimental Neuroimmunology 47, 50 (2018), filed as Ex. P15d on Sept. 30, 2020 (ECF No. 32-4) ("Kuwabara").

Ms. Sanchez, Dr. Nahm maintained, does not meet the GBS diagnostic criteria set forth under the Vaccine Injury Table. Nahm Third Rep. at 1–2. By contrast, in his view, her clinical symptoms and test results satisfy elements set forth for CIDP in a 2010 joint commission study aimed at improving the accuracy of CIDP diagnoses.[12] Tr. at 48; Nahm First Rep. at 22; P.Y.K. Van den Bergh et al., *European Federation of Neurological Societies/Peripheral Nerve Society Guideline on Management of Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Report of a Joint Task Force of The European Federation of Neurological Societies and The Peripheral Nerve Society — First Revision,* 18 Eur. J. Neurology 1, 10–11 (2011), filed as Ex. P15q on Sept. 30, 2020 (ECF No. 32-16) ("EFNS/PNS"). In particular, Petitioner's presentation was consistent with seven out of eight criteria (and only missed the final criterion because the necessary test was not performed).[13] Nahm First Rep. at 22. And the overall evidence that her

[12] Notably, Dr. Nahm stated that he would not use these criteria for diagnosing his own patients with CIDP but would use consensus guidelines instead. Tr. at 81–82. For a diagnosis of GBS, he would analyze the neurological symptoms, clinical history, and events prior to onset. *Id.*

[13] The first criterion was a progression over 60 days of the disease, which Ms. Sanchez exceeded. Tr. at 49. Second, weakness more than sensory symptoms. *Id*. Although Petitioner initially described sensory systems, she later described weakness greater than sensory loss, so he opined that she satisfied this second criteria. *Id*. Third, symmetric involvement of arms and legs, which Petitioner had both upper and lower extremity weakness. *Id.* Fourth, proximal muscles along with distal muscles, meaning the more proximal muscles (like the hip muscles) were affected along with the distal muscles (leg and foot muscles). *Id.* at 50. This matched Petitioner's complaints in the medical records. *Id.* Fifth, reduced deep tendon reflexes throughout, and although initially Dr. Nahm found one to two plus extremities in the upper extremities, eventually Petitioner lost her reflexes in the lower extremities and these symptoms waxed and waned over time. *Id.* Sixth, increased CSF protein without pleocytosis, which was present during her lumbar puncture. *Id.* Seventh, nerve conduction evidence of demyelination evidenced by Petitioner's NCS. *Id.* Finally, nerve biopsy showing segmental demyelination, which was not performed so it was unclear with Petitioner met this last criterion. *Id.*

symptoms progressed over time, rather than presented acutely then improved, was also more consistent with CIDP. Tr. at 52.

For additional support of the CIDP diagnosis, Dr. Nahm noted that two of the three neurologists who treated Ms. Sanchez had diagnosed her with CIDP. Nahm Third Rep. at 2. After Petitioner's EMG/NCS study in August 2016, two generally agreed that Petitioner's condition was CIDP, not GBS. Tr. at 46, 51. Dr. Nahm underscored Dr. Sharron's CIDP diagnosis in particular, since she had treated Petitioner for five years, while de-emphasizing the contrary view of Dr. Grogan, who only saw Petitioner a single time.[14] *Id.* at 84–85, 90, 129; Nahm First Rep. at 21. Dr. Nahm also pointed out that Dr. Grogan only said it was *possible* Petitioner had GBS, and also ultimately diagnosed her with some unspecified form of peripheral demyelinating neuropathy (which did not exclude CIDP). Tr. at 129, 133–34.

Dr. Nahm discussed giveway[15] weakness, which was mentioned by all three neurologists and could be evidence against CIDP as the proper diagnosis, but deemed it pejorative, adding that its acceptance could lead to the wrong diagnoses. Tr. at 79, 131. He pointed out that in some instances, like Petitioner's September 6, 2017 visit with Dr. Grogan, the overall exam was too limited to give the observation of giveway weakness much diagnostic weight. Tr. at 133; Ex. 7 at 6–8, 55–57. Dr. Nahm did allow for the possibility that Petitioner may have had some subjective symptoms, but nevertheless found in the record many documented instances of objective weakness (specifically in August and November of 2016). Tr. at 131–32, 135; Ex. 2 at 10; Ex. 7 at 42–44.

Petitioner's response to treatment was also consistent with the proposed CIDP diagnosis. Tr. at 46, 130–31; Nahm First Rep. at 23. As Dr. Nahm explained, one possible understanding of CIDP's pathogenesis is that it occurs due to the development of paranodal antibodies causing impaired nerve transmission. Treatment resistance or even clinical relapse following an initial response to IVIG has been associated with the presence of these autoantibodies. Nahm First Rep. at 23–24; A. Khoo, *Measuring Disease Activity and Predicting Response to Intravenous Immunoglobulin in Chronic Inflammatory Demyelinating Polyneuropathy*, Biomarker Res. 1, 7 (2019), filed as Ex. P15r on Sept. 30, 2020 (ECF No. 32-17). Here, the record established Petitioner's lack of response to later IVIG infusions, even though such treatment was at first ameliorative—and therefore this too suggested the presence of CIDP. Tr. at 46, 130–31; Nahm First Rep. at 23–24.

[14] Dr. Nahm also contested Dr. Grogan's opinion that Petitioner had GBS that later progressed into unrelated chronic pain disorders. In his first expert report, Dr. Nahm admitted that the medical records clearly showed Petitioner had some form of chronic pain disorder—either fibromyositis or fibromyalgia—prior to receiving the Tdap vaccine. However, while testifying, Dr. Nahm argued that he did not find anything in his review of the case to suggest that Petitioner *subsequently* had a chronic pain disorder like fibromyalgia. Tr. at 85.

[15] Dr. Nahm defined giveway to describe a lack of effort, or an impression by the examiner that the effort was poor. Tr. at 78–79.

Next, Dr. Nahm explained how the Tdap vaccine could theoretically cause CIDP.[16] Overall, he contended that "akin to molecular mimicry,"[17] the vaccine could invoke a cellular immune response in the body's T-helper cells to produce injury. Tr. at 54–55. Importantly, Dr. Nahm did not argue that any peptide sequence or structural homology between antigens in the Tdap vaccine and any nerve components could be demonstrated in this case. *Id.* at 65, 86. However, he proposed that the pertussis toxoid components of the Tdap vaccine could instigate a pathologic process later resulting in the demyelination characteristic of CIDP. Tr. at 65, 86, 92; Nahm First Rep. at 19. In particular, he maintained that the vaccine could upregulate certain CD4+ T-helper cells, which would in turn cause production of cytokines and other immune cells leading to a cross-reactive attack. Tr. at 92–93; Nahm Second Rep. at 3.

Thus, Dr. Nahm deemed his theory "akin to molecular mimicry," to the extent that in each case a vaccine component was the start of a pathologic process implicating the immune system. Tr. at 66, 87, 94. In effect, Dr. Nahm acknowledged, even if the antibody-driven humoral mechanisms[18] understood to drive GBS could not in this case be shown, it was equally likely that cellular mechanisms (here driven by T cells rather than B cell-produced antibodies) could lead to CIDP. Tr. at 58–60, 85–86; Nahm First Rep. at 18.

To support the theory, Dr. Nahm offered several items of medical literature. Tr. at 54–57; Nahm First Rep. at 20; P. Ross et al., *Relative Contribution of Th1 and Th17 Cells in Adaptive Immunity to Bordetella Pertussis: Towards the Rational Design of an Improved Acellular Pertussis Vaccine*, PLoS Pathogens 1, 4 (2013), filed as Ex. P15o on Sept. 30, 2020 (ECF No. 32-15) ("Ross"). Ross found evidence via an animal model that the acellular pertussis toxoid component in the Tdap vaccine could provoke the T helper 17 (Th17)[19] cells. Ross at 12–13. As a result, Ross's authors concluded that "Th17 cells mediate protective immunity" with respect to vaccines, like Tdap, that are intended to cause adaptive immunity to pertussis itself. *Id.* at 10.

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[16] Dr. Nahm admitted that this aspect of his opinion was not based on lab research he personally has performed with respect to immunological issues, but instead solely on literature research performed for this case, as well as the medical records at issue. Tr. at 91.

[17] Dr. Nahm defined molecular mimicry as a concept used in vaccine-related illnesses to explain how something in the vaccine can mimic the native antigens in the body, and the foreign antigens of the vaccine are then able to invoke an immune mechanism response. Tr. 53–54; Nahm First Rep. at 17–18. Dr. Nahm stated that although most of the associations are not present in larger studies, it is still the most widely cited explanation as to how vaccines cause injuries. Nahm First Rep. at 18; Y. Segal & Y. Shoenfeld, *Vaccine-Induced Autoimmunity: The Role of Molecular Mimicry and Immune Crossreaction*, Cellular & Molecular 586, 591–92 (2018), filed as Ex. P15e on Sept. 30, 2020 (ECF No. 32-5); M. Blank et al., *Autoimmunity: From Bench to Bedside* 1 (J-M Anaya et al. eds., 2013), filed as Ex. P15f on Sept. 30, 2020 (ECF No. 32-6).

[18] In so maintaining, Dr. Nahm admitted that while antiganglioside antibodies are thought to play an important role in the pathogenesis of GBS, the humoral factors in CIDP are much less clear. Nahm Second Rep. at 1.

[19] Dr. Nahm noted that he was specifically talking about the Th17 cells activation (which he specifies as a subset of CD4+ T cells), but not generally Th1 or 2 or A10. Tr. at 57; Nahm First Rep. at 20; Ross at 5.

Ross does not, however, purport to find that GBS, CIDP, or any other comparable inflammatory neuropathic condition, are *instigated* by these T-helper cells (let alone whether a vaccine could trigger the production of the T-helper cells in sufficient amounts to become pathogenic). Indeed, it says nothing at all about demyelinating disease. Dr. Nahm still deemed it significant, bridging its findings to other things understood about peripheral neuropathies. He thus noted (relying on a figure from another item of literature) that "T helper cells have already been implicated in the pathogenesis of CIDP." Nahm First Rep. at 20; E. Mathey et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: From Pathology to Phenotype,* J. Neurology Neurosurgery & Psychiatry 973, 975 (2014), filed as Ex. P15g on Sept. 30, 2020 (ECF No. 32-7) ("Mathey").

Mathey used a visual representation to reveal how different aspects of the immune response, including Th17 cells, would be implicated in CIDP's evolution, and Dr. Nahm maintained that these T-helper cells in particular created a ripple effect of cytokine and chemokine production, which could penetrate the blood-nerve barrier, adhering to different portions of the nerve to trigger an inflammatory response accounting for the symptoms of CIDP. Tr. at 64, 86; Nahm First Rep. at 18; Mathey at 975–76. Mathey, however (like Ross), does not contain the finding that these kinds of T-helper cells instigate CIDP, but rather observes they likely play some kind of assistive role "[d]uring active disease." Mathey at 976. Indeed, Mathey notes that other studies found more CD8+ T cells (the type of T cell more directly involved in attacking foreign antigens)[20] activated in CIDP than T-helper cells (although even the CD8+ T cells could not be confirmed to be drivers of disease as opposed to "evidence of a T cell response to chronic infection." *Id.* at 977. Thus, in no way does Mathey stand for the proposition that Th17 cells are central to CIDP's development.

Another item of literature considering the role different aspects of the immune system might play in CIDP was no different. J. Wolbert et al., *Deciphering Immune Mechanisms in Chronic Inflammatory Demyelinating Polyneuropathies*, JCI Insight 1, 4 (2020), filed as Ex. P21 on June 11, 2021 (ECF No. 42-4) ("Wolpert"). Wolpert, a review article, aimed to "synthesize recent advances in understanding CIDP pathogenesis," but came to no conclusions that would favor one aspect of the human immune response over another, in the way Dr. Nahm proposed. Wolpert at 1-2. Thus, Wolpert does allow that T-helper cells play *some* role in CIDP—and that the Th17 subtype, capable of production of cytokines likely significant to disease development, had been found in patients with CIDP. Wolpert at 4. But Wolpert's authors noted the need for additional study "to determine whether [the relevant cytokines] play critical roles in the immunopathology of CIDP." *Id.* at 5. Otherwise, Wolpert (like Mathey) noted that CD8+ T cells were often found in greater amounts in patients with CIDP, and that B cell-produced antibodies

[20] Mathey further elaborated that "[t]hese CD8+ T cell clones are enriched in the nerve suggesting than an antigen-driven, CD8+ cell mediated attack on the nerve contributes to the pathogenesis of CIDP." Mathey at 977.

also were likely significant (although their role could be variable, depending on the version of CIDP at issue). *Id.* at 5–6.

Dr. Nahm nevertheless contended that Th17 cells could upregulate in response to the pertussis toxoid component of the Tdap vaccine, promoting CIDP in turn. As further indirect proof, he made other observations. In particular, he noted that IVIG treatment could reduce these levels of immune cells—resulting in a corresponding cessation of the disease's progression, and hence in his view underscoring their significance to CIDP's pathogenesis. Tr. at 61.

In addition, Dr. Nahm offered some case reports of GBS and CIDP following Tdap vaccinations. Tr. at 66–69; Nahm First Rep. at 19; K. Kongbunkiat et al., *Clinical Manifestations and Outcomes of Guillain-Barré Syndrome After Diphtheria and Tetanus Vaccine (dT) During a Diphtheria Outbreak in Thailand: A Case Series*, Neurology Asia 149, 149 (2014), filed as Ex. P15j on Sept. 30, 2020 (ECF No. 32-10) (examining four cases of GBS after diphtheria and tetanus ("dT") vaccination during an outbreak in Thailand out of a total of 2,213,530 dT vaccines administered) ("Kongbunkiat"); H. Ammar, *Guillain-Barré Syndrome after Tetanus Toxoid, Reduced Diphtheria Toxoid and Acellular Pertussis Vaccine: A Case Report*, J. Med. Case Rep.'s 1, 2 (2011), filed as Ex. P15i on Sept. 30, 2020 (ECF No. 32-9) (finding an association between the Tdap vaccine and one patient subsequently diagnosed with GBS) ("Ammar"); N. Gregg et al., *Tdap Vaccination and Acute Demyelinating Events*, Neurology 1, 2 (2017), filed as Ex. P15k on Sept. 30, 2020 (ECF No. 32-11) ("Gregg") (reporting two cases where exposure to the Tdap vaccine result in acute demyelinating events); J. Pritchard et al., Risk of Relapse of Guillain-Barré Syndrome or Chronic Inflammatory Demyelinating Polyradiculoneuropathy Following Immunisation. J. Neurology Neurosurgery & Psychiatry 348, 348 (2002), filed as Ex. P15m on Sept. 30, 2020 (ECF No. 32-13) (analyzing the rate of relapse in a questionnaire of 1,100 GBS or CIDP patients after vaccination and finding that the greatest concern was 8.7 percent of patients had a relapse following a tetanus toxoid component) ("Pritchard").

Dr. Nahm also noted that an association between GBS and other vaccines is well known. Nahm First Rep. at 19; N. Principi & S. Esposito, *Vaccine-Preventable Diseases, Vaccines and Guillain-Barré Syndrome*, Vaccine 1, 2 (2019), filed as Ex. P15l on Sept. 30, 2020 (ECF No. 32-12) ("Principi"). And he discussed a 2012 Institute of Medicine ("IOM") report, which concluded that there was inadequate evidence to accept or reject a causal relationship between the diphtheria toxoid, tetanus toxoid, and acellular pertussis vaccines and CIDP. Tr. at 69–70; Nahm First Rep. at 19–20; Institute of Medicine, *Adverse Effects of Vaccines: Evidence and Causality* 1, 558–60 (Kathleen R. Stratton et al., eds., 2012) ("2012 IOM Report"). Because the 2012 IOM Report did not explicitly reject a connection, the possibility for an association remained.

Petitioner's medical history, Dr. Nahm maintained, was consistent with his causation theory. Petitioner had no prior symptoms of CIDP that predated her pregnancy, and she had no other significant pre-vaccination health occurrences that would explain her current condition. Tr.

at 39, 72–73; Nahm First Rep. at 23. He admitted, however, that the record did suggest some pre-vaccination symptoms that were arguably associated, or seen well after her purported April to May onset. Thus, Dr. Nahm agreed that Ms. Sanchez had experienced pain and significant issues at various times in 2015, including in association with chiropractic visits. Tr. at 75–76.[21] He pointed out, however, in response that the last chiropractic treatment had occurred six months prior to delivery, and there was never any bilateral numbness reported of the kind associated with GBS or CIDP. Tr. at 88. He also stated that although Petitioner had indicated intermittent hand numbness the year prior to her vaccination, there were no reports of any weakness or gait impairment until after vaccination. Nahm First Rep. at 24. But after vaccination on March 17, 2016, there were numerous records confirming the presence of neurologic symptoms associated with CIDP. Nahm First Rep. at 21; Ex. 1 at 24 (June 7, 2016 visit); Ex. 7 at 89-90 (June 21, 2016 visit) and 42-44 (August 18, 2016 EMG).

Finally, Dr. Nahm offered the opinion that the timeframe for Petitioner's symptoms onset was medically acceptable. He proposed that the evidence best supported an onset right around when Ms. Sanchez gave birth in April 2016, or 34-43[22] days after the March 17, 2016 vaccination, with May 3rd as the latest possible date. Tr. at 42–43, 44, 79–80, 89. He deemed this onset consistent with how long it would take for post-vaccination harm to manifest, pointing out that it was within the Table timeframe for a flu vaccine-GBS claim (although there is no comparable Table claim for CIDP after *any* vaccine). Such a timeframe (late April) was in his estimation consistent with his theory that the vaccination was the cause of Petitioner's condition because it's based on the onset of symptoms in a Table claim (six weeks). *Id.* at 44, 72, 83.

However, Dr. Nahm allowed in other regards that certain record evidence supported a far earlier onset. Thus, Dr. Nahm's initial expert report noted that Petitioner began to experience numbness, pain and weakness in her legs toward the *end* of her pregnancy, along with numbness in her hands. Nahm First Rep. at 26; Ex. 7 at 7. This might place onset toward the start of April. Then, on cross examination Dr. Nahm acknowledged that the medical record of Petitioner's June 7, 2016 visit to Dr. Bernstein placed her symptoms as having begun in *late May*, rather than the month prior. Tr. at 78; Ex. 1 at 24. He later admitted a tension between the fact that Dr. Sharron's

[21] Dr. Nahm was shown on cross examination records of Petitioner's multiple visits to the chiropractor, where Petitioner documented pain, and at one point during an April 23, 2015 visit, Petitioner noted left-hand numbness, weakness, and pain that was rated as a 10 out of 10 (although this visit was because of a car accident where Petitioner was rear-ended, which is not an injury related to vaccination). Tr. at 74-75; Ex. 5 at 11–12. During another chiropractic visit on July 6, 2015, Petitioner reported pain in her lower back and stiffness and tightness in her neck and a July 14, 2015 visit Petitioner reported tingling in her right knee, although again this was in reference to Petitioner's motor vehicle accident. Tr. at 75–76; Nahm First Rep. at 5; Ex. 5 at 6–7, 31.

[22] Dr. Nahm's testimony on this point is not wholly consistent with his written reports. The first expert report places onset around 30-42 days after vaccination (Nahm First Rep. at 24), while the second proposes it to have fallen 40-47 days after vaccination. Nahm Second Rep. at 4. The third report narrowed the timeframe to 42-49 days after vaccination (Nahm Third Rep. at 2), and relied on a 1979 study specific to GBS. *Id.*; L. Schonberger et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, Am. J. Epidemiology 105, 120–22 (1979), filed as Ex. P24 on Sept. 14, 2021 (ECF No. 53-2).

notes from the June 21, 2016 visit were detailed enough to help establish the kind of neurologic symptoms associated with CIDP—and yet those same records suggests a late-May onset. Tr. at 137–38. And he expressed bafflement at why the record from Petitioner's May 3, 2016 visit to Dr. Harden documented an absence of certain evidence of neurologic symptoms (tingling, weakness, or numbness), even though Petitioner testified she was at that time experiencing such problems, proposing in passing that it might simply reflect error or omission in the medical record. Tr. 76–77.

C. *Respondent's Expert - Brian Callaghan, M.D., M.S.*

Dr. Callaghan, a neuromuscular specialist in treatment of neuropathies like CIDP, prepared two written reports for Respondent and testified for Respondent in support of the contention that there is not a casual association between the Tdap vaccine and CIDP. *See generally* Tr. at 97–128. Report, dated March, 22, 2021, filed as Ex. A (ECF No. 37-1) ("Callaghan First Rep."); Report, dated July 26, 2021, filed as Ex. C (ECF No. 49-1) ("Callaghan Second Rep.").

Dr. Callaghan received his undergraduate degree from the University of Michigan, his medical degree from the University of Pennsylvania in 2004, and his Masters in Science from the University of Michigan in 2011. CV, filed as Exhibit B on March 26, 2021 (ECF No. 37-6) ("Callaghan CV") at 1; Tr. at 98. He became a clinical lecturer at the University of Michigan Health System's Department of Neurology in 2009 and has been an Associate Professor of Neurology there since 2018. Callaghan CV at 1; Tr. at 98–99. He is licensed to practice medicine in Michigan, and he is board certified by the American Board of Psychiatry and Neurology and the American Board of Electrodiagnostic Medicine. Callaghan CV at 1; Tr. at 98. He sees about 500 patients a year. Tr. at 99. Dr. Callaghan has published 120 articles and medical book chapters, about 75 percent of which focus on neuropathies, and his research interest lies in diagnostic evaluation and testing of peripheral neuropathies. Callaghan CV at 10–17; Tr. at 99. Dr. Callaghan has averred that he treats approximately 30 patients with CIDP per year. Callaghan First Rep. at 1; Callaghan Second Rep. at 1.

The first feature of Dr. Callaghan's overall opinion was his diagnostic contention that Petitioner more likely suffered from a resolved case of GBS, with her subsequent symptoms and treatment pertaining to pre-vaccination conditions that did not constitute GBS sequelae. Tr. at 100–01, 124; Callaghan First Rep. at 4. Dr. Callaghan agreed with Dr. Nahm that it can be difficult to diagnose GBS or CIDP correctly in its initial stages, requiring careful consideration of a patient's overall clinical history. Tr. at 101, 109–110. The need to take a broader view of Petitioner's history was thus especially important in this case, given the GBS/CIDP overlap.[23] *Id.* at 109–10.

[23] Although Dr. Callaghan opined in his written reports that Petitioner developed either GBS or CIDP, he clarified during his testimony that it was in his view much more likely that Petitioner has GBS (even though he acknowledged he could not rule out CIDP with any degree of medical certainty). Tr. at 115–16. Callaghan First Rep. at 6; Callaghan Second Rep. at 2.

Looking generally at the record, Dr. Callaghan observed many factors that he deemed consistent with GBS. For example, he noted that Petitioner had presented with neurologic symptoms over the course of only a few weeks before reaching her likely nadir. Tr. at 101, 109–10. Petitioner did later improve, even though she maintained some persistent symptoms. *Id.* at 101. In addition, the three treating neurologists she saw all documented instances of giveway weakness,[24] rather than true/demonstrable weakness. *Id.* at 101, 103–04, 110, 122; Ex. 7 at 6, 85; Ex. 9 at 29. The existence of such giveway weakness suggested to him that Petitioner had recovered from GBS sometime thereafter (although a specific timeframe was not specified), but still had a lot of symptoms of pain, numbness, and weakness—all of which were consistent to her symptoms prior to vaccination. Tr. at 125.

Dr. Callaghan further noted that at least one of Petitioner's treaters, Dr. Grogan, had proposed GBS as the proper diagnosis, distinguishing her other remaining symptoms as reflective of her underlying conditions of fibromyalgia, fatigue, and chronic pain syndrome. Tr. at 101, 118, 125; Ex. 7 at 6–8. In so emphasizing, he noted that in his experience it was common for patients with GBS who had continued symptoms related to preexisting conditions to have their diagnosis incorrectly changed to CIDP, simply because some seemingly-associated symptoms persisted, and offered literature in support of the view that CIDP could easily be misdiagnosed. Tr. at 101, 117, 126.[25]

At the same time, however, Dr. Callaghan acknowledged that Dr. Grogan (whom Petitioner saw only a single time) was in the minority of treaters herein disagreeing with the CIDP diagnosis.[26] Tr. at 116. But he noted that it was not, in his experience, uncommon for patients to seek second and third opinions in such contexts, and also to prefer the determinations of treaters with whom they agreed. *Id.* at 116–17. The very fact that Petitioner did have multiple neurologists underscored the degree to which the etiology of her symptoms was unclear. *Id.* at 116.

Another factor Dr. Callaghan felt supported a GBS diagnosis was Petitioner's response to treatment. Tr. at 101–02. Ms. Sanchez had initially had a positive response to IVIG, discontinuing it only due to its side effects (i.e., rash). *Id.* at 101–102, 108–09, 119, 126. She thus did not receive it consistently enough to deem it ineffective—and in any event even Petitioner herself seemed to acknowledge that her symptoms were more broad and acute. *Id.* at 22, 101–102, 108–09, 119, 126.

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[24] Dr. Callaghan defined giveway weakness as a lack of true weakness, possibly due to a patient embellishing or not putting enough effort. It is a way for a physician to indicate in a medical record that the patient is not fully cooperating with the treater exam. Tr. at 102, 118.

[25] R. Lewis, *Chronic Inflammatory Demyelinating Polyneuropathy: Etiology, Clinical Features, and Diagnosis*, UpToDate 1, 1–12 (2011), filed as Ex. P15q on June 11, 2021 (ECF No. 42-6) ("Lewis"). But although Lewis discussed classification, pathogenesis, epidemiology, clinical manifestations, and diagnostic criteria for CIDP, it does not in fact appear to conclude that half of all CIDP patients are misdiagnosed.

[26] Dr. Callaghan in fact contested whether all other neurologists that Petitioner saw in fact embraced the CIDP diagnosis. Tr. at 122.

But CIDP patients, in Dr. Callaghan's experience, would usually prefer to maintain treatment due to their chronic symptoms. *Id.* at 119.

The steroidal treatment Petitioner received, and her response to it, was in Dr. Callaghan's view further evidence of why GBS was the better diagnostic conclusion. As a general matter, he proposed, steroids are ineffective in treating GBS but effective for patients with CIDP—and Ms. Sanchez did not see a benefit from the steroids she received. Tr. at 126–27. However, Dr. Callaghan acknowledged as well that Petitioner did not receive steroids consistently enough to ascertain whether it was in fact ameliorative of her symptoms—although he felt this meant that the effectiveness of the treatment in CIDP generally could not be determined from Petitioner's history. *Id.* at 127.[27]

The second half of Dr. Callaghan's opinion was devoted to contesting the existence of a causal association between the Tdap vaccine and CIDP. Tr. at 103; Callaghan First Rep. at 4. He agreed that some versions of the flu vaccine[28] were reasonably associated with GBS. Callaghan First Rep. at 5. But he denied that convincing evidence supported the same association with the Tdap vaccine. Callaghan First Rep. at 5. And nothing offered by Dr. Nahm to support causation was deemed persuasive by Dr. Callaghan. Dr. Nahm had admitted that he could not demonstrate homology between antigenic components of the Tdap vaccine and amino acid sequences in the protein components of nerve structures, thus ruling out molecular mimicry as an applicable mechanistic theory. Callaghan Second Rep. at 1, 5. At most, Dr. Nahm had reliably established that a pertussis infection (as opposed to the vaccine) could stimulate one part of the immune system associated with CIDP's pathogenesis—but not that the stimulated T-helper cells responding to an active pertussis infection *were causal* of CIDP. Tr. at 103.

In so maintaining, Dr. Callaghan questioned the extent to which the literature filed by Dr. Nahm was supportive of his causation theory. Tr. at 107, 110. Mathey, for example, did effectively describe many of the pathophysiologic features of CIDP and the parts of the immune system implicated in it—including two kinds of T cells, macrophages, and antibodies. Tr. at 104; Mathey at 975–76. But in Dr. Callaghan's reading, the main takeaway from Mathey was that CIDP's pathophysiology was diverse, with numerous "unknowns" about specifics in terms of triggers or driving mechanistic factors. Tr. at 105. Dr. Callaghan also noted that Mathey specifically acknowledged that there were no known infectious triggers for CIDP—diminishing the

[27] Dr. Callaghan also pointed out that there are placebo effects of IVIG and steroid treatment, and in IVIG specifically, causing virtually any person with comparable symptoms to feel initially better. Tr. at 127. Indeed, the fact that IVIG can suppress inflammation means it often feels beneficial to patients—even when the true cause of their condition is not likely immune-mediated. *Id.* at 127–28.

[28] Dr. Callaghan qualified this assertion, however, noting that (as reflected in literature offered by Dr. Nahm) it might be the case that only a 1976 version of the flu vaccine was in fact reasonably associated with GBS. Callaghan First Rep. at 5; Principi at 2.

persuasiveness of a contention that *any* associated vaccine could be causal.[29] Tr. at 104–05; Mathey at 978.

Dr. Callaghan also discussed the case reports cited by Dr. Nahm. As a general matter, he observed that case reports were a kind of evidence valuable in flagging areas for future study, but were otherwise anecdotal and not able to establish causation in any reliable sense. Callaghan First Rep. at 5. At most, case report evidence could only establish a temporal association between CIDP and vaccination. *Id.* And the case reports filed herein were distinguishable for other reasons. Kongbunkiat and Ammar, for example, both discussed only GBS. Tr. at 106–07; Kongbunkiat at 149; Ammar at 2. Gregg focused on central nervous system demyelinating disorders, which Dr. Callaghan argued was too broad a category to have relevance to CIDP, which was a distinguishable peripheral neuropathy. Tr. at 106–07. Gregg at 2. And Pritchard had several methodological weaknesses, including self-identified CIDP patients, an extremely small sample size, and reliance on patient reports for relapse rather than verifiable evidence of them.[30] Callaghan First Rep. at 4; Pritchard at 349. Pritchard also only addressed relapse of CIDP after vaccination and not onset. Callaghan First Rep. at 4.

Regarding the 2012 IOM report, Dr. Callaghan did not agree with Dr. Nahm's conclusion that because it did not formally find evidence of a *lack* of a causal relationship that it left the door open to an association. Tr. at 104; Callaghan First Rep. at 5. Not only was it difficult generally to "prove a negative" (i.e., prove that no association was possible), but the 2012 IOM report specifically noted that there was insufficient evidence to find *anything* (either epidemiologically or from a mechanistic basis) that could show an association either way. Tr. at 104, 123; Callaghan First Rep. at 5; 2012 IOM Rep. at 558–60. Thus, the 2012 IOM Report determination was less supportive of Petitioner's causation theory than alleged.

Other literature, however, rebutted Petitioner's causation contentions in Dr. Callaghan's estimation. One specific article looked at possible antecedent events that might causally explain CIDP but reached conclusions contrary to Dr. Nahm. Callaghan First Rep. at 4; P. Doneddu et al., *Risk Factors for Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP)): Antecedent Events, Lifestyle and Dietary Habits. Data from Italian CIDP Database,* Eur. J. of Neurol. 1, 1–2 (2019), filed as Ex. A, Tab 1 on Mar. 26, 2021 (ECF No. 37-2) ("Doneddu"). Doneddu investigated dietary and lifestyle differences between CIDP patients and their partners, using a questionnaire to evaluate whether a number of events prior to onset, including vaccination, had occurred. Doneddu at 1–2. Of the 411 patients studied, Doneddu found only 1.5 percent had

[29] Dr. Callaghan more broadly questioned Dr. Nahm's conclusion that any kind of environmental antigenic stimulus could explain CIDP. Nahm First Rep. at 17, 19; Callaghan First Rep. at 5.

[30] Dr. Callaghan also noted that recall bias is common in studies that ask about exposures in those with known disease. Callaghan First Rep. at 4.

received any vaccines in the six weeks preceding diagnosis. *Id.* at 3. The authors concluded that the studied antecedent events were unlikely to play a role in the risk of CIDP. *Id.* at 6.

Petitioner's medical history was, in Dr. Callaghan's reading, unsupportive of the conclusion that the Tdap vaccine had caused her neurologic injury. Tr. at 101. Prior to vaccination, Petitioner had several established preexisting conditions, such as fibromyalgia, fatigue, and cervical radiculopathy, none of which could by themselves be attributed to a vaccine (or the one at issue), and as already noted Dr. Callaghan concluded that many of Petitioner's later symptoms were not sequelae of GBS (his preferred diagnosis) but instead reflected these antecedent underlying problems. *Id.* at 100. In addition, none of Petitioner's treating physicians linked the Tdap vaccine to Petitioner's injury. *Id.* at 109. And Dr. Callaghan did not recall an instance from his own practice in which a patient that received the Tdap vaccine subsequently suffered from CIDP (although he admitted that he had treated patients with GBS where a vaccine was a potential cause of their condition). *Id.* at 109, 115.

Dr. Callaghan's opinion also included consideration of Petitioner's onset and its relationship to causation. He maintained that the records best supported the conclusion that her neurologic symptoms began in late May 2016.[31] Tr. at 107. This established an onset of symptoms eight weeks after vaccination, which Dr. Callaghan deemed too distant from vaccination to be reasonably associated. *Id.*; Callaghan First Rep. at 5–6. He relied on GBS-oriented evidence for this conclusion. Thus, according to a 2004 IOM Report, an association between the 1976-77 swine flu vaccination and GBS could be concluded if onset fell in as late as six to eight weeks post-vaccination, although the strength of the association fell as time passed. Callaghan First Rep. at 5–6; Institute of Medicine, *Immunization Safety Review: Influenza Vaccines and Neurologic Complications* 1, 72 (Kathleen R. Stratton et al., eds., 2004) at 48–49 ("2004 IOM Report"). Here, because Petitioner's symptoms started eight weeks after vaccination, the timeframe was too attenuated to support an association. Callaghan First Rep. at 5–6.

III. Procedural History

After the case's initiation in July 2018, Petitioner filed medical records supporting the claim, and then Respondent's Rule 4(c) Report was filed on March 29, 2019 (ECF No. 13), with a more substantive amended version filed two months later. ECF No. 14. On September 24, 2019, this case was selected for ADR as part of a since-discontinued program. ECF No. 16. But such mediation efforts were unsuccessful. ECF No. 25. Accordingly, after the case was put back on a

[31] In support, Dr. Callaghan specifically cited to Petitioner's June 7, 2016 visit, in which she reported that her symptoms began three weeks prior. Tr. at 107–08, 122; Callaghan Second Rep. at 2; Ex. 1 at 24. He also noted a January 24, 2018 visit where she indicated her symptoms started two weeks after delivering her baby. Tr. at 108; Ex. 9 at 16. Other, more temporally-proximate visits reinforced the likelihood that onset began in late May. Tr. at 120-21; Ex. 9 at 89 (June 21, 2016 visit).

litigation track, the parties began filing expert reports, concluding the process in the summer of 2021. Hearing of the matter occurred on September 15, 2021. ECF No. 39.

IV. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[32] In this case, Petitioner does not assert a Table claim.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs*., 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause

[32] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245.

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) ("[h]owever, in the past we have made clear that simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof" (citing *Moberly*, 592 F.3d at 1322)). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a

'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained

in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be

more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed

every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. An Overview of Relevant Medical Terms and Applicable Prior Decisions

Dr. Nahm has reasonably defined CIDP as an immune-mediated demyelinating neuropathy that affects both large and small fiber peripheral nerves, resulting in symptoms of numbness, tingling, weakness, imbalance, loss of coordination and pain. Nahm First Rep. at 17; Kuwabara at 47. CIDP shares similar characteristics to GBS, since both are immune-mediated demyelinating peripheral neuropathies. But although Dr. Nahm maintained that CIDP is nothing more than a chronic form of GBS, there are key differences in their clinical presentation that distinguish the two—and they also cannot be assumed to have the same pathogenic mechanisms. Nahm First Rep. at 16–17. CIDP progresses over a longer period of time than GBS, which features acute weakness within one to two weeks after onset. Hanewinckel at 365. In addition, little is known about CIDP's most likely causes, triggers, or pathogenesis, in comparison to GBS (where a variety of specific infectious triggers have been identified, as well as the situs of cross-reactive autoimmune attack). Tr. at 104-05; Nahm First Rep. at 17, 19; Mathey at 973, 978 ("for the majority of patients the specific target of the autoantibody response is unknown"). Thus, although many Program decisions seem to have assumed that what is known about GBS applies fully to CIDP given their similarities, this assumption is not well founded.

There are ample prior decisions associating different vaccines with CIDP (more often than not, the flu vaccine), and petitioners have settled many such cases on favorable terms.[33] *See Jastisan v. Sec'y of Health & Hum. Servs.*, No. 13-937V, 2016 WL 4761950 (Fed. Cl. Spec. Mstr. Aug. 10, 2016). I have myself acknowledged their existence in my own prior decisions, and the fact that such determinations should be given *some* consideration as persuasive guidance. *See, e.g., Mason v. Sec'y of Health & Hum. Servs.,* No. 17-1383V, 2022 WL 600415, at *21 (Fed. Cl. Spec.

[33] Of course, prior decisions from different cases do not control the outcome herein (and this goes double for cases that are settled, and hence resolved without a reasoned determination). *Boatmon*, 941 F.3d at 1358–59; *Hanlon v. Sec'y of Health & Hum. Servs*., 40 Fed. Cl. 625, 630 (1998). But special masters are empowered to draw upon their experience in resolving Vaccine Act claims. *Doe v. Sec'y of Health & Hum. Servs*., 76 Fed. Cl. 328, 338–39 (2007) ("[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is most probative of a claim") (emphasis added). They would therefore be remiss in ignoring prior cases presenting similar theories or factual circumstances, along with the reasoning employed in reaching such decisions.

Mstr. Feb. 4, 2022); *Houston v. Sec'y of Health & Hum. Servs.*, No. 18-420V, 2021 WL 4259012, at *16 (Fed. Cl. Aug. 19, 2021); *Strong v. Sec'y of Health & Hum. Servs.*, No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018).

However, I have identified no recent[34] *reasoned* decisions in which a special master explained how or why the Tdap vaccine was likely causal of the claimant's CIDP. *See Houston*, 2021 WL 4259012, at *17–18 ("determining that the first *Althen* prong was not met as "[p]etitioner has made no showing comparable to what would be required to prove that [an] association [between Tdap and GBS] translates to an association between Tdap and CIDP"). And there are several decisions in the past ten years that suggest the strength of a vaccine association with CIDP is far weaker than what may have previously been presumed. In a 2014 case, for example, a petitioner was unsuccessful in claiming her ongoing neurological condition was aggravated by two influenza vaccinations. *Jacunksi v. Sec'y of Health & Hum. Servs.*, No. 09-524V, 2014 WL 5168422, at *7 (Fed. Cl. Spec. Mstr. Sept. 23, 2014). The special master highlighted an IOM report (among other things) which specifically found insufficient available evidence to support an association between influenza vaccine and CIDP. *Id.* at *14.

II. Petitioner Has Preponderantly Established His CIDP Diagnosis

It is often appropriate for a special master to first determine which alleged injury is best supported by the evidence before applying the *Althen* test—particularly when the injury is disputed—so that "the special master could subsequently determine causation relative to the injury." *Broekelschen*, 618 F.3d at 1346. In some cases, determining the injury obviates entirely the need for any *Althen* analysis, since the petitioner's claim, and causation theory, is dependent on a finding of a specific injury. *Id.*

In this case, the parties dispute the proper diagnosis—and indeed it is the case that Petitioner's claim relies on a determination that she likely suffered from CIDP post-vaccination. Respondent argues that she actually suffered from GBS. The record best supports Petitioner's contention, for several reasons.

First, there is ample, trustworthy treater support for Petitioner's preferred diagnosis. Two neurologists—including Dr. Sharron, who had treated Petitioner for five years and was the first neurologist to see her—embraced CIDP as the diagnosis, while only one (Dr. Grogan, whom it appears she saw only a single time) preferred GBS or something comparable. Although I am never bound to accept a treater's opinion, I may give weight to their views. *Snyder*, 88 Fed. Cl. at 746

[34] In a case that is now 17 years old, the Court granted a motion for review reversing a special master's determination that a tetanus toxoid-containing vaccine had not been shown to cause CIDP. *Kelley v. Sec'y of Health & Hum. Servs.*, 68 Fed. Cl. 84 (Fed. Cl. 2005). *Kelley,* however, is distinguishable legally as well as factually. In particular, *Kelley* relied on older literature that seemed to assume that CIDP and GBS were two sides of the same coin—even though core literature discussing CIDP filed in this case does not so conclude. Thus, although I am not in any event bound by the Court's findings in *Kelley,* I deem it to be based on now-superseded science and medical views, and do not afford it persuasive value in deciding this case.

n.67. Here, treaters Dr. Sharron and Dr. Bhavaraju-Sanka reached conclusions that seem reasonable, based upon the records in which they are set forth. Second, the medical records largely appear consistent with the conclusion that Petitioner was suffering from CIDP. Petitioner underwent complete diagnostic workups, including labs, MRIs, a lumbar puncture, and EMG/NCS tests. This is the evidence upon which the treaters relied. Dr. Nahm also persuasively established that the majority of the EFNS/PNS criteria for CIDP were met.

Dr. Callaghan makes a number of reasonable points supporting his contrary diagnostic view. There is evidence that at times Petitioner's weakness could not be documented, due to her occasions of "giveway" conduct when examined, allowing for less certainty that the weakness was neurologic in nature. It is also true that many of her post-vaccination symptoms seem consistent with the kinds of pain and other concerns she unquestionably experienced pre-vaccination, and thus her ongoing symptoms could be reflective of those conditions rather than evidence of CIDP. And the kinds of treatment response to IVIG and steroids (with the inferences they provide in suggesting whether a patient is experiencing the chronic symptoms associated with CIDP or not) cannot be fully evaluated, since Petitioner did not undergo these treatments long enough. These factors, plus Dr. Callaghan's personal experience, do cast some doubt on the CIDP diagnosis. Nevertheless, the overall record preponderates in Petitioner's favor on this issue.

III. Petitioner Has not Carried Her Burden of Proof

A. *Althen Prong One*

Petitioner has not established that the Tdap vaccine could cause CIDP, because Dr. Nahm's opinion was insufficiently medically/scientifically reliable. He tried to analogize his theory of causation to one (very common in Program cases) based on molecular mimicry as a mechanism. Yet, in the same breath he admitted that not only could he not establish the kind of homology between vaccine antigens and self structures in the nerves that is a common starting point for this theory when offered, but moreover that mimicry between components of the vaccine and a proposed myelin target (resulting in production of autoantibodies that would cross-react against the myelin by mistake) *was not the mechanism* he was relying upon at all. Tr. 53–54; Nahm First Rep. at 20. He also pointed to no specific antibody created in response to the Tdap vaccine that might cross-react against nerve myelin, in the manner GBS is believed to progress.

Accordingly, the fact that molecular mimicry is often embraced to explain how autoimmune pathologic processes might unfold[35] is of no help to Petitioner in this case. Nor could

[35] In fact, as I have observed in prior cases, even when molecular mimicry *is* a plausible basis for a causation theory, more must be done to preponderantly establish causation than to simply assert that it is an oft-accepted mechanism in Program cases. *See, e.g.*, *McKown v. Sec'y of Health & Hum. Servs.,* No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (citing *Devonshire v. Sec'y of Health & Hum. Servs.,* No. 99-031V, 2006 WL 2970418, at *15 (Fed. Cl. Spec. Mstr. Sept. 2006)) ("[b]ut merely chanting the magic words 'molecular mimicry' in a Vaccine Act case does not render a causation theory scientifically reliable, absent *additional evidence* specifically tying the

Petitioner simply rely on science applicable to GBS (and in turn the causation theories that have associated it with certain vaccines). Despite the overlap between GBS and CIDP, far less is known about CIDP and its overall pathogenesis. And this case does not in any event involve the flu vaccine—the vaccine best associated, from a scientific/medical perspective, with GBS or (arguably) other peripheral neuropathies involving demyelination.[36]

Petitioner therefore needed something else to link the Tdap vaccine to the relevant injury. Dr. Nahm attempted to provide that link by focusing on a putative association between the pertussis component of the vaccine and certain T-helper cells he maintained had something to do with CIDP's progression and course. Certainly he offered reliable literature, like Mathey and Ross, establishing that these immune cells promote a healthy adaptive response to pertussis vaccines. Similarly, Wolpert does establish that T-helper cells have some role in CIDP's pathogenesis—although perhaps of less-significant role that primary T cells actually responsible for autoimmune damage. But as discussed above (and pointed out by Dr. Callaghan, as well), these items of literature do not find that this class of T cells is central to, or instigative of, CIPD's course. Nor has it been reliably established that the pertussis toxoid component invariably causes upregulation of these immune cells to the degree necessary to spark disease *at the outset* (as opposed to at some later point in CIDP's progression—past when vaccination would no longer be a factor in impelling a disease process). Dr. Nahm's contention that the T-helper cells created a ripple effect of cytokine and chemokine production, which got through the blood nerve barrier was also without corroborative support.

Thus, to support his claim, Dr. Nahm (who could reference no specific expertise from his past studying CIDP or vaccines that might be causal of it) mostly relied on independent literature that described what is known generally about immune processes implicated in CIDP—but those same items, viewed collectively, did not preponderantly establish likely causation. He also relied heavily on case reports—a class of evidence not typically given substantial weight in the Program. *See K.O. v. Sec'y of Health & Hum. Servs*., No. 13-472V, 2016 WL 7634491, at *11–12 (Fed. Cl. Spec. Mstr. July 7, 2016) (discussing appellate precedent on case reports). And Dr. Callaghan persuasively explained why the case reports offered were all distinguishable. Moreover, the 2012 IOM Report may not have excluded causation as a possibility, but it certainly does not *support* causation either, and thus provides faint assistance to Petitioner in carrying her preponderant burden.

This is not a situation where Respondent's expert rebutted Petitioner's showing in every regard. Dr. Callaghan relied heavily on his personal sense that the Tdap-CIDP association was not

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mechanism to the injury and/or vaccine in question") (emphasis in original), *mot. for review den'd,* 76 Fed. Cl. 452 (2007)).

[36] By contrast, in cases involving the flu vaccine, and where some effort was made to identify a potential autoantibody and situs for cross-reactive attack, I have been willing to find the "can cause" prong was met (although not so robustly that it could not be rebutted). *See Mason,* 2022 WL 600415, at *27.

accepted in his field—a conclusion deserving of limited weight, even if medical/scientific community acceptance sheds a little light on the connection between a vaccine and injury. He also cited some items of literature, like Doneddu, that I have noted in other cases lack persuasive power. *See, e.g., Houston*, 2021 WL 4259012, at *18 ("cited for the proposition that "antecedent events" like vaccination were not likely causal—since all [Doneddu's] authors observed was that a *small percentage* of CIDP patients in the study had been vaccinated before onset. (More troubling for Petitioner, however, if the fact that none of these subjects received the Tdap vaccine)").

But Petitioner's "can cause" showing fails nevertheless—because it was not preponderantly carried in the first place. The filed literature, coupled with Dr. Nahm's opinions and testimony, do not establish it is "more likely than not" that the pertussis component of the Tdap vaccine is sufficient to stimulate an autoimmune process due to T-helper cell upregulation and leading to the initiation of CIDP.

B. *Althen Prong Two*

The record also does not permit the conclusion that the Tdap vaccine likely "did cause" Ms. Sanchez to experience CIDP. No treaters ever proposed any association between the March 2016 vaccination and Petitioner's subsequent diagnosis. Nor is there any record evidence of any reaction to the vaccine that might suggest an aberrant immune process had begun. There is only the fact that at some time after receipt of the vaccine in March 2016—a month to six weeks, depending on when onset began (a matter separately discussed below)—Petitioner's neurologic symptoms began. This temporal association alone cannot establish a causal relationship. *See Capizzano*, 440 F.3d at 1327.

I note as well that Petitioner's medical record *does* include ample evidence of pre-existing symptoms that *seem* neurologic in some respects. In particular, she had a history of weakness, pain (even on one recorded occasion reaching a 10 out of 10 on the pain scale), left-hand numbness, tingling in her right knee, lower back and stiffness, and tightness in her neck—all in the year prior to vaccination. She also had treatment specific to a car accident in this period. Additionally, Petitioner's symptoms began close-in-time to the completion of her pregnancy. Dr. Callaghan's observation that some of her lingering symptoms seem less reflective of continuing CIDP than those she first reported in 2016 also is entitled to some small weight. All of the above suggests a milieu in which a variety of a causal factors independent of vaccination could have played a role in causing a neurologic injury.

In the end, I cannot find on this record that these intervening/intercurrent factors better explain her injury (and as already noted, I have found it more likely than not that she was properly diagnosed with CIDP in any event). But this kind of record *nevertheless* impedes the success of Petitioner's prong two showing, since these contemporaneous factors have not been fully addressed in Dr. Nahm's opinion. Untangling such neurologic, or neurologic-like symptoms is

extremely difficult based on this record—which only further diminishes (if weakly) the likelihood that vaccination was in this case causal.

C. *Althen Prong Three*

The experts disagreed on a precise onset date, with Dr. Nahm favoring a six-week post-vaccination onset[37] while Dr. Callaghan proposing eight weeks. Notably, however, *both* opinions suffered from the same error, in that they were rooted in science pertaining specifically to GBS (as well, in Dr. Nahm's case, as the Table establishes a 3 to 42-day period for a flu-GBS claim). *See* §§ 300aa-13(a)(1)(A), 11(c)(1); § 300aa-14(a), as amended by 42 C.F.R § 100.3(a)(XIV)(D); 300aa-11(c)(1)(C)(ii)(I).[38] This is not a GBS case, however (nor is the flu vaccine at all implicated), so relying on what is known about the timeframe involving a distinguishable illness (an acutely-presenting condition that does not typically wax and wane, as is true of CIDP) and vaccine is unhelpful. *See Mason*, 2022 WL 600415, at *25 ("what is known about GBS's onset timeframe cannot simply be borrowed as a template to understand a likely onset timeframe for CIDP").

Nevertheless (and despite the fact that little is understood about the possible environmental triggers for CIDP), literature offered in this case and prior cases support the medical acceptability of onset of symptoms within the timeframes proposed by *either* expert. 2004 IOM Report at 48–49; *see Daily v. Sec'y of Health & Hum. Servs.*, No. 07-173V, 2011 WL 2174535, at *9 (Fed. Cl. May 11, 2011) (finding that onset of CIDP within a few weeks of vaccination was a medically acceptable timeframe). And in prior cases I have only rejected vaccine-CIDP timeframes that were substantially longer. *See e.g., Patel v. Sec'y of Health & Hum. Servs.*, No. 16-848V, 2020 WL 2954950, at *18-21 (Fed. Cl. Spec. Mstr. May 1, 2020) (stating that seven months for vaccine-caused CIDP too long); *Strong*, 2018 WL 1125666, at *21 (noting that four months between flu vaccine and onset of CIDP was too long).

As a result, I find that Petitioner's neurologic symptoms onset more likely than not occurred in a medically-acceptable timeframe, measured from her March 2016 vaccination. But because I have found that the Tdap vaccine has not been shown herein to likely cause CIDP, Petitioner's satisfaction of this single *Althen* prong does not alter my determination.

[37] As noted previously, Dr. Nahm's timeframe opinion was refined as he filed expert reports. Tr. at 42–43; Nahm First Rep. at 24; Nahm Second Rep. at 4; Nahm Third Rep. at 2.

[38] Claimants cannot "piggyback" on the Table requirements when attempting to prove a non-Table claim. *See Greene v. Sec'y of Health & Hum. Servs.*, No. 11-631V, 2018 WL 3238611, at *9 (Fed. Cl. Spec. Mstr. May 7, 2018) (noting that an expert's opinion on the timing issue of a brachial neuritis claim relied on conclusory determinations that the "Table time periods were not that far off the time period in question (something Program law says is not permitted)"), *dismissing petitioner's claim*, 2019 WL 4072110 (Fed. Cl. Spec. Mstr. Aug. 2, 2019), *aff'd*, 146 Fed. Cl. 655 (2020), *aff'd*, 841 F. App'x 195 (Fed. Cir. 2020).

CONCLUSION

It cannot be assumed that because GBS is closely associated with the flu vaccine, that *any* related condition is likely similarly attributable to *other* vaccines. Rather, claimants must do the "heavy lifting" imposed upon them in causation-in-fact cases and show how the vaccine in question could cause a different condition. *Lampe*, 219 F.3d at 1360. What is known about the related condition and vaccine may well supply a useful "roadmap," but in the end the claimant's showing must reliably establish causation.

This has not been accomplished in this case, as the Tdap vaccine has not been shown to likely lead to CIDP. For this reason, I am compelled to dismiss this claim.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[39]

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[39] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.